Nitric Oxide Genotoxicity Protection by *Ginkgo biloba* Extract

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Over the last years, the *Ginkgo biloba* plant has grown a considerably good reputation within the scientific community and today its potential in terms of beneficial effects is well sustained in the literature [1]. Most of the studies involving the use of plant material, however, only face their antioxidant properties and just a little was explored relatively to their antigenotoxic activity.

The excessive production of oxidative species, being reactive oxygen species (ROS) or reactive nitrogen species (RNS), can be stressful to the cell to a point where they compromise survival. In between their molecular targets, ROS can affect DNA, possibly causing the loss of its stability and integrity, imposing a very dangerous threat. NO is recognized for its biological roles in the regulation of vasodilation, and nervous system and immune system signaling as well as for its potentially adverse effects [2]. Depending on the molecules NO encounters inside the cell, it may oxidize into peroxynitrite or dinitrogen trioxide (among many others), both molecules being able to interact with and modify DNA [3].

The chemical analysis of the ethanolic *Ginkgo biloba* extract (GBE), revealed the presence of some characteristic compounds.. Also, a protective effect against SNP was observed in viability assays, in which fission yeast *Schizosaccharomyces pombe* wild type strains and DNA repair-affected mutants were used. Cell cycle analysis revealed that incubation with GBE alone causes a quicker advance in cell cycle progression and that treatment with GBE slightly reduces the delay caused by SNP. Finally, experiments involving the oxidative stress response protein Pap1 fused with GFP pointed to a possible protection mechanism, where cell interaction with the extract may be functioning as a mild stress elicitor, preparing cells for the stress induced by NO. Putting all the evidence together, GBE protects cells from the effect of SNP through a DNA-repair independent mechanism, which may involve the scavenging of NO and subsequent decrease in DNA modifications, and/or the signalling of oxidative stress-response proteins preventing the excessive accumulation of oxidant molecules.

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